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DIETARY INTAKE OF CAROTENE IN NONSMOKERS WITH AND WITHOUT PASSIVE SMOKING AT HOME

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There is considerable epidemiologic evidence suggesting an inverse relation between dietary intake or blood levels of beta-carotene (provitamin A) and cancer, especially lung cancer (1–6). Several studies have demonstrated that cigarette smokers have lower blood beta-carotene or carotenoid levels than nonsmokers (7–10). Therefore, beta-carotene status as measured by dietary intake or blood levels may be a confounding factor in the relation of cigarette smoking to lung cancer.

Passive smoking has been linked with an increased risk of lung cancer in nonsmokers in a number of studies (11–17). To our knowledge, there have been no studies examining the relation of beta-carotene status to passive smoking. We therefore conducted a cross-sectional study of current dietary carotene intake in a population of 2,142 nonsmokers for whom we collected information about passive smoking at home.

METHODS

Study population

The study population consisted of the 2,142 nonsmokers in the group of 3,899 Kaiser Permanente Medical Care Program members who received multiphasic health checkups in Oakland, California, during 1985 and who satisfactorily completed self-administered research questionnaires regarding tobacco use, alcohol consumption, and diet. The membership of the Kaiser Permanente Medical Care Program, which provides health care to approximately 25 per cent of the population of the San Francisco Bay Area, is heterogeneous (18). Those who take multiphasic health checkups are also heterogeneous but tend to be better educated and more health-conscious (19–20). Excluded from the study population were 766 nonsmokers who did not complete all the questions on the dietary questionnaire.

Assessment of passive smoking

Passive smoking at home was ascertained by the response to the question on the tobacco use questionnaire, "On the average, how many hours per week are you exposed to cigarette, cigar or pipe smoke in your home because of smoking by others? (Write in 000 if less than 1 hour per week) ——— hours." This questionnaire was administered to Kaiser Permanente multiphasic health checkup examinees between 1979 and 1986; a study of passive smoking based on the questionnaire has previously been published (21). Nonsmokers were classified as being exposed to passive smoke at home if the response to this question was greater than zero.

Assessment of dietary carotene intake

A 30-item semiquantitative food frequency questionnaire for the assessment of

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carotene (carotenoid precursors of vitamin A, e.g., beta-carotene, alpha-carotene, lycopene), retinol (preformed vitamin A), and total vitamin A intake was modified from a more extensive food frequency questionnaire designed by Willett et al. (22). A carotene value was assigned for an average serving size of each item, and the value was multiplied by the number of servings reported per week to obtain the weekly carotene intake. Precise estimates of specific carotenoids were not possible based on available food composition tables. The values derived from published food tables represent most of the beta-carotene plus about half the alpha-carotene and a small fraction of lycopene (10, 23).

Statistical methods

All statistical analyses were performed with SAS programs (24). *T* tests were used to assess the statistical significance of differences between means, and linear regression models were used to perform trend tests and multivariate analyses. In the multiple linear regression models, age and weight were entered as continuous variables while other characteristics were entered as indicator variables, as follows: sex; race (white, black, Hispanic, Asian, other); education (highest grade completed: 0–9, 10–12, technical/business, partial college, college graduate, post graduate, unknown); alcohol intake (nondrinker, former drinker, current drinker of less than 1, 1–2, 3–5, or 6+ drinks/day).

RESULTS

Baseline characteristics of nonsmokers exposed and those unexposed to passive smoke at home

Overall, 356 (16.6 per cent) persons of the study population reported passive smoking at home ("the exposed") (table 1). The exposed had a lower mean age and higher proportions of females, blacks, and current alcohol consumers than the unexposed. Educational status and mean body mass index were similar in the two subgroups.

TABLE 1

Baseline characteristics of nonsmokers exposed and those unexposed to passive smoke at home

Characteristic	Unexposed	Exposed
Total no.	1,786	356
Age*	42.9 ± 15.6	37.4 ± 13.7
Age range (years) (%)		
<35	36.7	49.2
35–54	36.3	37.1
≥55	27.0	13.8
Sex (% female)	59.1	68.8
Race (%)		
White	59.9	48.0
Black	18.6	33.1
All others	21.5	18.9
Education (%)		
≤High school	15.1	13.8
>High school	67.8	67.4
Unknown	17.1	18.1
Body mass index*	25.0 ± 4.8	25.9 ± 5.1
Alcohol consumption (%)		
Never and past	19.5	15.4
Current	79.8	84.3
Unknown	0.7	0.3

* Mean ± 1 standard deviation.

Dietary carotene intake in nonsmokers exposed and those unexposed to passive smoke at home

There were consistently higher mean carotene intakes in the unexposed across the sociodemographic and alcohol consumption categories examined (table 2). Most of the differences were statistically significant ($p < 0.05$). The linear regression model coefficient for passive smoking at home in relation to carotene intake (−1,185) was highly significant ($p = 0.007$), confirming the association between passive smoking at home and a lower dietary intake of carotene.

Mean dietary carotene intake by quartile of weekly duration of passive smoking at home in the exposed nonsmokers

Dietary intake of carotene was somewhat higher for the lower two than for the upper two quartiles of passive smoking duration (table 3). However, there was no statisti-

TABLE 2

Mean dietary carotene intake (IU/day) in nonsmokers exposed and those unexposed to smoke at home, by various characteristics, and difference between means

Characteristic	Unexposed	Exposed	Difference	p^*
Total population	8,697	6,793	1,904	0.0001
Sex				
Male	8,108	6,530	1,578	0.02
Female	9,105	6,912	2,193	0.0001
Age (years)				
<35	7,374	6,144	1,230	0.03
35–54	8,786	7,035	1,751	0.009
≥55	10,370	8,458	1,912	0.10
Race				
White	8,949	6,995	1,954	0.0006
Black	8,241	6,043	2,198	0.006
All others	8,398	7,674	724	0.47
Education				
≤High school	8,682	7,158	1,524	0.22
>High school	8,680	6,900	1,780	0.0002
Unknown	8,774	6,143	2,631	0.007
Alcohol consumption				
Never and past	9,686	6,214	3,472	0.004
Current	8,429	6,917	1,512	0.005

* p value associated with the t test of the difference of the means.

TABLE 3

Mean dietary carotene intake by quartile of weekly duration of passive smoking at home in exposed nonsmokers

Quartile	Hours/week	Beta-carotene intake (IU/day)
1 (lowest)	≤2	6,718
2	3–7	8,108
3	8–24	6,032
4 (highest)	≥25	6,474

cally significant graded relation between carotene intake and duration of passive smoking either by a trend test on the unadjusted data ($p = 0.30$) or by multiple linear regression analysis of data with adjustment for age, sex, race, body weight, educational status, and alcohol intake ($p = 0.51$).

DISCUSSION

Mean dietary carotene intake was found to be lower in nonsmokers exposed to passive smoke at home than in nonsmokers who were not exposed to passive smoke at home, indicating that dietary beta-carotene intake is a potential confounder in studies of the relation between passive smoking and lung cancer. The relative risk of lung cancer associated with cigarette smoking is so great that it is unlikely to be attributable to confounding by beta-carotene intake, since the protective effects that have been associated with beta-carotene intake tend to be much lower (2, 6). However, the relative risk for lung cancer associated with passive smoking has averaged about 2 in several studies, so that evaluation of a potential confounder such as beta-carotene is important in attempting to elucidate causality (11–17). In order to assess the extent of potential confounding quantitatively, we simulated it with artificial data, assuming that a decrease in carotene intake equivalent to the difference in carotene intake between the middle of the highest and the lowest quartiles of carotene risk (approximately 2 standard deviations based on our study population) would double the risk of lung cancer. We found that an observed relative risk of 2 for passive smoking and lung cancer would be reduced to a true relative risk of approximately 1.8 with consideration of dietary carotene intake, suggesting that failure to adjust for intake of dietary carotene may inflate estimates of

the risk of passive smoking, and its statistical significance, by amounts that are modest, yet noteworthy. Only one study of which we are aware has controlled for dietary beta-carotene intake in the analysis of the passive smoking-lung cancer relation, and this did not eliminate a positive association between passive smoking and lung cancer (17). We know of no studies that have controlled for blood levels of carotenoids in the analysis of this relation, nor do we have data regarding such blood levels in this study population.

The most likely explanation for this finding lies in important lifestyle differences between nonsmokers exposed and nonsmokers unexposed to passive smoke at home; this is supported by two differences in other characteristics. The first is the higher proportion of current alcohol consumers in the exposed subgroup, and the second is the slightly higher mean body mass index of this subgroup despite its considerably lower mean age. Although nonsmokers as a whole tend to have higher dietary intake or blood levels of carotene than cigarette smokers (7–10), it is possible that within households, the dietary preferences of nonsmokers are similar to or influenced by those of smokers, at least in regard to foods containing carotene. An alternative but, in our view, less likely hypothesis is that passive smoke exposure affects the appetite of the nonsmoker, resulting in decreased carotene intake.

We also examined dietary retinol intake. Although passive smoking at home was associated with a somewhat lower dietary retinol intake, the difference was not statistically significant ($p = 0.38$ with adjustment for age, sex, race, education, weight, and alcohol intake). The lack of a significant difference for retinol intake indicates that the intake of meat products, which are the primary dietary source of retinol, are similar in the two groups. In addition, we examined the dietary intake of carotene in relation to reported passive smoking in other settings, i.e., in small (e.g. airplane, office, car) and large (e.g. restaurant, hotel lobby, lecture hall) spaces. The results were similar to those for passive smoking at home. Nonsmokers exposed to passive smoking in small and in large spaces had lower carotene intake than nonsmokers not exposed to passive smoke in these settings ($p = 0.02$ for small spaces and 0.05 for large spaces, with the same adjustments as for passive smoking at home).

In summary, the mean dietary intake of carotene was lower in nonsmokers exposed to passive smoke at home than in nonsmokers not exposed to passive smoke at home, and the difference was statistically significant after controlling for age, sex, race, educational status, body weight, and alcohol intake by multiple linear regression. We conclude that dietary beta-carotene intake is a potential confounder and should be measured whenever possible in studies of the relation between passive smoking and lung cancer.

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